Chromosomal excision of a new pathogenicity island modulates Salmonella virulence in vivo

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Abstract

Although the excision of unstable pathogenicity islands is a phenomenon that has been described for several virulent bacteria, whether this process directly affects the capacity of these microorganisms to cause disease in their hosts remains unknown. Salmonella enterica serovar Enteritidis (S. Enteritidis) is an enterobacterium that harbors several unstable pathogenicity islands that can excise from the main bacterial chromosome. Here we have evaluated whether excision of one of these pathogenicity islands, denominated as Region of Difference 21 (ROD21), is required for S. Enteritidis to cause disease in the host. By means of genetic targeting of the integrase encoded by the ROD21 we have generated S. Enteritidis strains unable to excise ROD21. The failure to excise ROD21 significantly reduced the capacity to cause a lethal disease and to colonize the spleen and liver of mice, as compared to wild type S. Enteritidis. On the contrary, S. Enteritidis strains overexpressing an excisionase protein increased the frequency of ROD21 excision and showed an improved capacity to cause lethal disease in mice. Accordingly, strains unable to excise ROD21 showed an altered expression of genes located in this pathogenicity island. Our results suggest that the genetic excision of the pathogenicity island ROD21 in S. Enteritidis modulates the capacity of this bacterium to cause disease in mice due to a change in the expression of virulence genes.

Keywords

Salmonella enterica serovar enteritidis, pathogenicity island excision, systemic infection